which were of normal morphology to disintegrate during the coagulation process. Thus no thromboplastic factor was released and no prothrombin consumed.

Freezing and thawing was shown to promote the prothrombin consumption of normal native platelet rich plasma as well as that of the patient. The experiment corroborates the observation made by others that part only of the platelets disintegrates during coagulation. The descriptive name cocciarrhectic thrombocytopathy is proposed for the condition characterized by normal platelet morphology, normal platelet thromboplastic factor content and impaired prothrombin consumption.

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A NEW TREATMENT FOR RESISTANT PRESSURE SORES

J. T. HAUCH, *Toronto*

IN ALL THE CASES described below, standard hospital methods of treating pressure sores (antiseptic compresses or ointments or systemic antibiotics) were used and either left the condition unchanged or were attended by deepening or spreading of the ulcer.

Because vitamin E in large doses systemically and, as an ointment, locally has been alleged to improve ulcers for gangrenous patches resistant to therapy, it was decided to try this in the present cases.

Vitamin E was first used in Case 1 (J.L.) more as a counsel of despair than with any expectation that it would improve a condition with an otherwise hopeless outlook. To assess the value of vitamin E ointment locally, only one of the three sores in this case was at first treated with it. It soon became apparent that this ulcer was healing much faster than the others and after a while all ulcers were given the benefit of local as well as general vitamin E therapy. The remarkable improvement in the condition of the ulcers, and in the patient's general condition, led us to repeat the treatment in the other three cases, and in each the results obtained have been judged to warrant further use of the method.

These cases are reported here because we believe that use of vitamin E locally and systemically deserves trial in larger series by others confronted with the same problem of the resistant ulcer that refuses to heal.

CASE 1.—J.L., aged 26, was admitted to St. Joseph's Hospital on August 24, 1955, with a five-year history of symptoms of disseminated sclerosis. On admission she had symptoms and signs of advanced multiple sclerosis with flexion contractures of legs and poor general condition. There were no demonstrable areas of superficial anæsthesia, and the patient was quite susceptible to pain stimuli, with a moderate degree of reduction in hot and cold sensation and light touch. She had no control of either bladder or rectum. There was a marked flexure deformity of the lower extremities, with the legs flexed on the thighs to about an angle of 15° at the knees. The thighs were flexed on the abdomen to an angle of about 40° at the inguinal region. A definite coarse tremor of both the upper and lower extremities was noted on any movement, whether voluntary or passive. There was also a marked clonus of both knees and ankles. At the same time there was fairly good motor use of the upper extremities but very little motor power in the lower extremities. All deep reflexes were exaggerated, there was bilateral plantar extension, and abdominal reflexes were absent. There was no aphasia, and the patient was quite intelligent, sensible and co-operative.

In October 1955 she first developed a pressure sore on her sacral area, and soon afterwards the skin over both hips became affected. She was treated at first with saline compresses and then with powdered yeast applied locally. In January 1956 the bedsores were irradiated with ultraviolet light. Other measures included the application of Hygeol compresses. There was no response to any of the treatments used; the ulcers became larger and the patient became extremely depressed while her physical condition deteriorated.

On June 7, 1956, when four large areas of deep ulceration and necrosis were present, a trial was begun of vitamin E by mouth and local application to *one* of the areas. Hygeol compresses or cicatrin powder was used as control on the other three areas. Three weeks after the start of vitamin E therapy, a stricter control of local treatment was begun by applying to the three control areas only the Vaseline base used as a vehicle for the vitamin E. The oral dosage of vitamin E was at first 800 units a day (200 i.u. q.i.d.), but after three weeks this was increased to 1600 units (400 i.u. q.i.d.).

The condition of the ulcers at the beginning of vitamin E treatment and during the latter was as follows:

Right Hip. There was an ulcer measuring 11.7 x 6.8 cm., with a depth of 0.3 cm. Most of its base was necrotic but not, apparently, secondarily infected.

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Left Hip. Two ulcerated areas were present, an upper measuring 9.2 x 7.1 x 0.6 cm., with a necrotic base, and a lower measuring 12.5 x 6.3 x 0.5 cm. and of similar appearance.

Back. There was a large ulcer 17.8 x 7.6 x 0.8 cm., with a necrotic, dirty-looking base from which, however, no pathogenic micro-organisms could be cultured.

It was soon apparent that the bedsore to which vitamin E ointment was applied was healing, whereas progress in the other three areas was very slow, though present after the oral administration of vitamin E was begun. Hence, after six weeks, the ulcers on the right hip were also given topical therapy on July 19, and by September 22 we were so convinced of the superiority of combined topical and oral vitamin E over simple oral therapy that we began its application to all ulcerated areas, 8-hourly.

Motor power was practically normal in shoulder muscles. Grasp in both hands was poor but there was some power in the muscles of the upper arm and forearm. Response to pin-prick was absent on the medial aspect of both forearms. There was very slight sensation from the umbilicus down and in the lower extremities.

Reflexes:	*Left*	*and*	*Right*
Abdominal	absent		absent
Triceps			
Biceps	+++		+++
Radial	+++		+++
Knee	++		++
Ankle	++		++
Babinski			

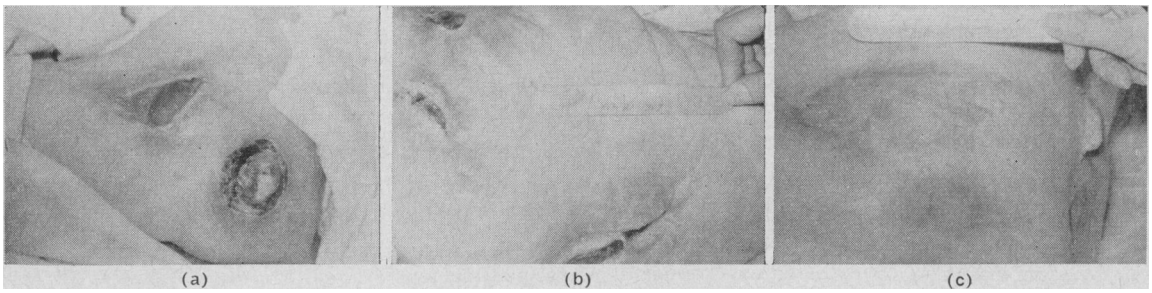


Fig. 1 (a).—J.L. Large deep pressure sore on buttock; more superficial sore over lumbosacral area. (b) Feb. 21, 1957. All pressure sores much smaller, granulating well. (c) May 16, 1957. All pressure sores practically healed.

The upper area on the left hip (given vitamin E topically from the start) healed over very well, but unfortunately when the patient was allowed to start sitting up in a chair, it began to break down again and she had to be returned to bed. There is now an ulcerated central area 3.7 x 2.4 mm., with healthy granulations level with the skin. Of the lower left area, only a slit 2.1 x 2 cm. remains and it is granulating well. Even after one week of topical vitamin E the area on the right hip showed signs of granulation and of growth of healthy-looking skin. This area is now completely healed, and vitamin E ointment has been discontinued. The area on the back was necrotic until vitamin E ointment was applied; it then rapidly began to fill in with healthy-looking granulations while normal skin began to grow in. By October 29 the area was almost entirely covered with new skin. The patient was then permitted to sit up (for the first time in 14 months), but on November 8 she was put back in bed because several of the areas had begun to break down, apparently because of the trauma inflicted in movement from bed to chair. At present, she is left with two shallow ulcerated areas measuring 5.6 x 3.5 and 2.9 x 0.9 cm., respectively; the intervening and surrounding skin looks quite healthy.

CASE 2.—J.McG., aged 18 years, was admitted to St. Joseph's Hospital on May 30, 1956, in an unconscious state after an accident in which he had sustained a fracture dislocation of the 6th and 7th cervical spines with partial hemisection of the spinal cord. He was at first not expected to live, but actually made steady progress.

After recovery of consciousness, he was found to have anæsthesia from just below the clavicle involving the rest of the body. Motor function was normal to the 6th cervical segment on the right and 8th cervical on the left. All lower limb reflexes were absent. The patient was put on a fracture board with traction and there was no appreciable change in his condition up to June 27, 1956. On June 30, examination revealed the following findings.

The findings on August 30 were essentially the same as on June 30.

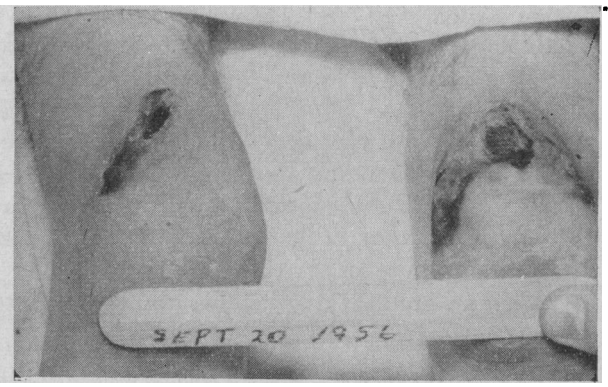
On August 27, pressure sores in anæsthetic areas were observed just over the sacral area and on both heels. The ulcerated area on the back measured 7.6 x 6.9 cm. at its maximum development and was about 1 cm. deep; its base was necrotic and edges were undermined. The areas on the heels were fairly superficial and attained a maximum of 2.8 x 1.7 and 2.2 x 1.4 cm., respectively. They did not respond to routine treatment with Hygeol, etc.

Vitamin E treatment (400 mg. q.i.d. orally) was started on September 21, and at the same time local application of vitamin E ointment was begun. Whereas the heel ulcers responded well and were completely healed by November 23, after two months' treatment, the necrotic area on the back showed very poor response in the first four weeks. At this time it was realized that the patient had not been receiving vitamin E locally with regularity but had been dressed with Vaseline gauze owing to the staff's failure to appreciate the importance of local plus general treatment. When this error was corrected, and a spray of vitamin E solution was applied to the undermined edges, progress was marked. The undermined areas filled in and the edges began to show signs of new skin formation. At the same time, healthy granulations began to fill the cavity. The ulcer now measures 6.6 x 5.2 cm. The cavity has filled in with granulations to skin level, except on the right where there is an area 0.4 cm. below skin level.

CASE 3.—Mrs. A.B., aged 78, was admitted on June 12, 1956, because of diabetes mellitus with obesity, diabetic neuritis and gangrene of the right big toe. Her toe was amputated on July 19; she was put on a 1000-calorie diet, and her diabetes seemed well controlled on 35 units of protamine zinc insulin each morning. Because of her obesity, the patient was difficult to manage and showed no desire to move about, spending most of her time lying on her back.

On August 26, the sacral area first showed signs of breaking down. The patient was put on Furacin oint-

Fig. 2 (a)



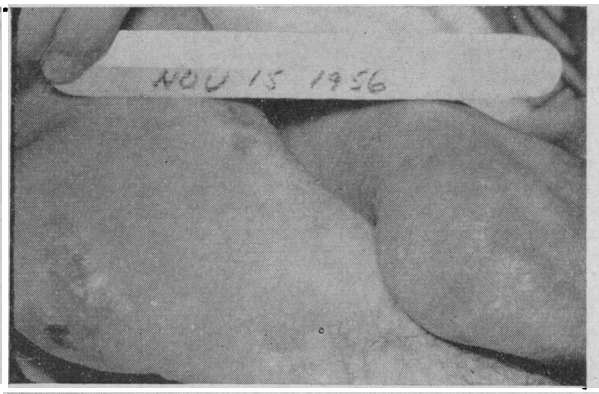
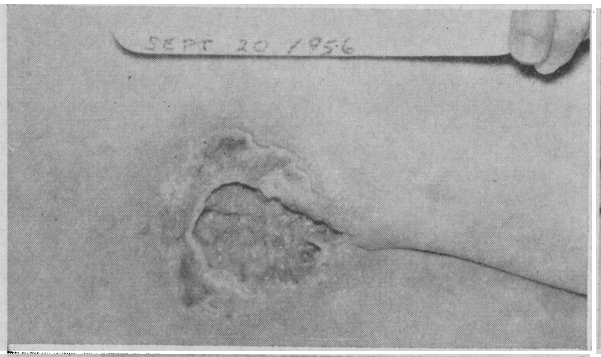


Fig. 2 (b)

Fig. 3 (a)



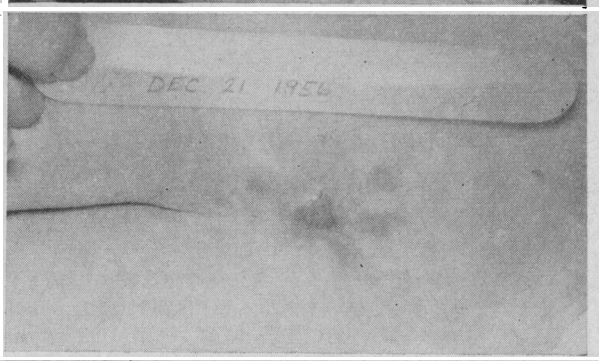
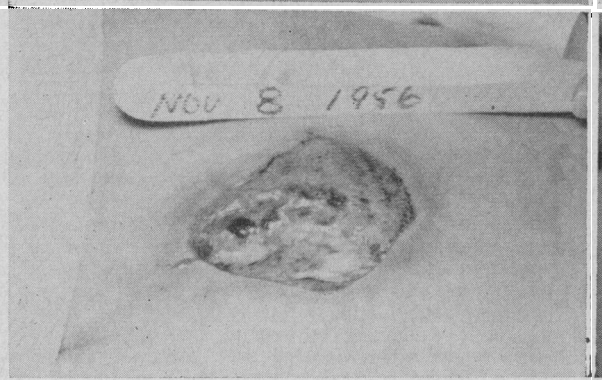


Fig. 3 (b)

Fig. 4 (a)



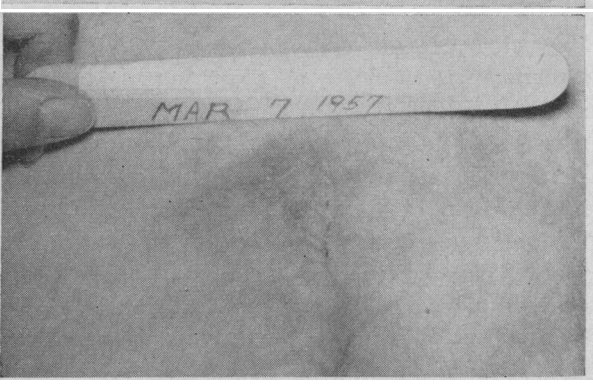


Fig. 4 (b)

Fig. 2 (a).—J.McG. September 20, 1956. Extensive and deep pressure sores on both heels.
Fig. 2 (b).—Nov. 15, 1956. Areas practically healed.
Fig. 3 (a).—Mrs. B. Sept. 20, 1956. Extensive deep pressure sore at base of sacrum.
Fig. 3 (b).—Dec. 21, 1956. Only small superficial area remaining unhealed.
Fig. 4 (a).—Mrs. A. Nov. 8, 1956. Sacral pressure sore.
Fig. 4 (b).—March 7, 1957. Sore completely healed.

ment and Dicrysticin, but the ulcer became deeper and larger. On August 31, treatment was changed to Hygeol compresses, and a week later it was again changed to irrigation with Hygeol and application of chloramphenicol ointment. However, the ulcer continued to increase in size and depth until September 21, when vitamin E therapy was started (400 mg. q.i.d. by mouth; spray to undermine edges and to sinus; half-strength ointment to ulcer).

At the start of vitamin E therapy the ulcer (6.7 x 3.9 cm. and 2.2 cm. deep) was covered in most areas by a thick tenacious tissue which appeared necrotic and gangrenous in parts. The borders of the ulcer were undermined by necrotic tissue, and there was a sinus running laterally and coming to the surface at a distance of 10 cm. from the main ulcer. Much of the necrotic tissue was cut away before vitamin E therapy was started. Ten days later the area appeared much healthier, with healthy granulation tissue. The one area still containing thick tenacious material was excised, and the use of full-strength vitamin E begun. Two weeks later the sinus had completely healed and its exit was epithelized. The main ulcer had filled in remarkably well and the undermined skin was completely filled in. In another week the area was almost completely covered in by healthy-looking skin, and the patient was shortly thereafter discharged home because she was becoming depressed over her long hospital stay. She now has a small granulating area the size of a pea which has required cauterization down to skin level.

CASE 4.—A.A., a woman 71 years old, was admitted to hospital on October 9, 1956, with a fracture of the neck of the left femur. On October 30, a large sacral ulcer appeared and was treated with Hygeol and liquid paraffin compresses. Three days later, slough was excised and the treatment continued. On November 9, because there was no improvement, therapy was changed to vitamin E, 400 mg. q.i.d. by mouth and applied locally to the ulcer 8-hourly as half-strength ointment.

At this time, the ulcer measured 7.0 x 6.8 cm. and was 0.5 cm. in depth. The whole area was covered with a dirty-looking slough from which no organisms were grown on culture, but which appeared gangrenous at one

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point. On November 30, when the vitamin E ointment was increased to full strength, the ulcer base appeared quite healthy with good granulations, and the sides were beginning to fill in with healthy-looking skin.

On December 15, the ulcer measured only 5.5 x 1.4 cm., with a depth of only 0.1 cm. Granulations were all healthy and there was no evidence of any degenerating tissue. This case again presented the problem of management of a woman who would not help herself and spent most of her time either lying or sitting down, with the added factor of frequent urinary incontinence.

The author expresses his thanks to Webber Pharmaceuticals Limited, Toronto, Canada, for the generous supplies of Vita-E Succinate, Vita-E Ointment and Vita-E Spray used in this study.

WESTERN EQUINE ENCEPHALOMYELITIS: REPORT OF A CASE IN MONTREAL

V. PAVILANIS, M.D.,*
ISOBEL L. WRIGHT, M.D.† and
M. SILVERBERG, M.D., C.M.,‡ *Montreal*

WESTERN equine encephalomyelitis (W.E.E.) has been recognized as occurring in humans since 1938 when Beatrice Howitt of the University of California isolated the virus from the brain of a 20-month-old boy who had died on the fifth day of his acute illness.[16] An encephalitis had been observed in horses and mules for many years in the U.S.A. but it was not until the summers of 1930 and 1931, during two epidemics in California, that a virus was demonstrated and isolated as the etiological agent through the studies of Meyer, Hering and Howitt. The possibility of this virus causing disease in humans was suggested at that time by Meyer.[15]

In Canada, the first recognized epizootic in horses occurred in Saskatchewan in the summer of 1935. This was followed by more severe outbreaks in 1937 and 1938 which included the Province of Manitoba. In the wake of this equine infection a number of humans developed an unidentified disease of the central nervous system which at first was thought to be non-paralytic poliomyelitis. This was later established as W.E.E. from two human cases by Fulton and Burton in 1939.[3]

*Chief of the Virus Department, Institute of Microbiology and Hygiene of the University of Montreal.
†The Department of Pædiatrics, The Montreal Children's Hospital, and McGill University.
‡The Montreal Children's Hospital and McGill University. Present address: Children's Medical Center, Boston, Mass.

The most extensive human epidemic occurred in 1941, involving the Provinces of Saskatchewan and Manitoba, and the states of North and South Dakota, Minnesota, Montana and Nebraska. There was a milder epidemic in 1947 and since that time the whole west has been plagued with endemic infection. As there have been no reports of human W.E.E. in Canada, east of Manitoba, it was thought important to report an atypical case of this disease occurring in Montreal.

D.A., a 20-month-old male infant, had been attending the allergy clinic of the Montreal Children's Hospital for treatment of recurrent attacks of asthma for six months previous to the present illness, which began on September 17, 1955. On that day he developed a hoarse voice, a dry hacking cough and dyspnœa. At 2 a.m. on September 20, because of increasing dyspnœa and restlessness he was taken to the out-patient department where on examination he was thought to have an acute laryngitis or epiglottitis with an allergic component. He was treated with intramuscular chloramphenicol 500 mg., a subcutaneous injection of 0.5 c.c. of adrenalin (epinephrine) 1 in 1000, ½ of a 0.25 g. aminophylline rectal suppository to be continued q.6.h., Franol (ephedrine, luminal, theophylline) ½ tablet q.i.d. and Elixir of Pyribenzamine (tripelennamine) 10 mg. q.i.d. By 10 a.m. the same day on his return to the clinic, he was much improved but by 10 p.m. his respiratory rate was again very rapid with dyspnœa and pallor; he was again brought back to the hospital and admitted the night of September 20. When seen on the ward he was very pale, showed marked respiratory difficulty with supraclavicular, intercostal and subcostal indrawing, and was thrashing about his bed making purposeless movements. His temperature was 102° F., his heart rate was 200 per minute and his respiratory rate was 60. The remainder of the physical examination was done with considerable difficulty and was non-contributory. He was placed in an oxygen croupette, given intramuscular chloramphenicol and streptomycin, and the aminophylline and Pyribenzamine were to be continued. The department of otolaryngology was consulted and on their advice emergency tracheotomy was postponed. Eight hours after admission his respiratory distress had improved but his peculiar maniacal behaviour persisted. He had no other abnormal signs referable to the central nervous system apart from an equivocal nuchal rigidity. The optic discs appeared normal. The nurses noted on two or three occasions that he had tremors of his upper extremities. Sedation with phenobarbital was unsuccessful but was finally achieved on his third hospital day with chlorpromazine. His temperature ranged between 101° and 103° F. until September 25, when it remained at 100° F. for 48 hours before it subsided to normal on September 29. He showed definite signs of improvement by the end of the week, and by his tenth hospital day he was playing like any healthy child of comparable age.

Laboratory investigation.—Hæmoglobin value 8.6 g. %, white cell count 11,200 with 6200 neutrophils, 4400 lymphocytes and 800 monocytes. There was no stippling of the red cells. Urinalysis was normal. Intradermal tuberculin and blood Wassermann tests were negative. Blood calcium, potassium, sodium, chlorides, CO_2 combining power, sugar and non-protein nitrogen were within normal limits on September 22. Cerebrospinal fluid (CSF) samples obtained on September 21 and 26 were clear and colourless with only two cells and a negative Pandy test in each case. The protein, sugar and chlorides of the CSF of September 26 were within the normal range. Roentgenological examination of the